

# Predicting 1-year mortality after elective abdominal aortic aneurysm repair

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**Objective:** Benefit of prophylactic abdominal aortic aneurysm (AAA) repair requires sufficient survival to overcome operative risk. Since death within 1 year of elective open or endovascular (EVAR) infrarenal AAA repair likely indicates ineffective treatment, we developed a prediction model for 1-year mortality to aid clinical decision-making.

**Methods:** We used a prospective registry of 1387 consecutive patients who had undergone elective AAA repair from 2003 to 2007 by 50 surgeons from 11 hospitals in Northern New England. Cox proportional hazards were used to analyze potential risk factors for 1-year mortality, including medical comorbidities, aortic clamp site, preoperative risk factor modification (eg, beta-blockade), and aneurysm diameter.

**Results:** Thirty-day and 1-year mortality after open repair (n = 748) was 2.3% and 5.8%, and after EVAR (n = 639) was 0.5% and 5.7%, respectively. Factors associated with death within 1-year after open repair were: age  $\geq 70$  ( $P = .007$ ; hazard ratio [HR] 2.9, 95% confidence interval [CI] 1.3-6.3), history of chronic obstructive pulmonary disease (COPD) ( $P < .0001$ ; HR 3.6, 95% CI 1.9-7.0), chronic renal insufficiency (creatinine  $\geq 1.8$ ) ( $P = .008$ ; HR 2.8, 95% CI 1.3-6.2) and suprarenal aortic clamp site ( $P < .0001$ ; HR 3.8, 95% CI 1.9-7.5). Depending on the number of risk factors present, predicted 1-year mortality after open repair varied from 1% in patients with no risk factors to 67% in patients with four risk factors. Our model demonstrated excellent correlation between observed and expected deaths ( $r = 0.97$ ). For EVAR, identified risk factors for death within 1 year included a history of congestive heart failure (CHF) ( $P = .002$ ; HR 3.2, 95% CI 1.6-6.5), and aneurysm diameter  $\geq 6.5$  cm ( $P = .04$ ; 95% CI 1.0-4.8). Depending on the number of risk factors present, predicted mortality ranged from 3.6% to 23%. A model using CHF and aneurysm diameter correlated well with actual mortality rates, with an observed to expected ratio of 0.96.

**Conclusion:** Predictors of 1-year mortality can identify patients less likely to benefit from elective AAA repair. These factors differ for open repair vs EVAR and should be considered in individual patient decision-making. Our EVAR model had less impact on 1-year survival, even if CHF and large AAA diameter were present. However, a combination of age, COPD, renal insufficiency, and need for suprarenal clamping have significant impact on 1-year mortality after open AAA repair. Consideration of these variables should assist decision-making for elective AAA repair, especially in borderline cases. (J Vasc Surg 2009;49:838-44.)

Elective abdominal aortic aneurysm (AAA) repair is a prophylactic operation designed to improve survival by preventing aneurysm rupture. Decision-making requires a careful analysis of rupture risk vs operative risk, but also of life expectancy, since patients must live long enough for the benefit of surgical repair to overcome operative risk. Prediction of survival after AAA repair has historically focused on the early postoperative (in-hospital or 30-day) or long-term (5-year) intervals.<sup>1-9</sup> In this regard, early survival demonstrates the safety of AAA repair, but does not convey late benefit. Similarly, 5-year survival is determined by

many other causes of late mortality, principally cardiac disease, stroke, and malignancy, and is difficult to predict on an individual basis. Clinical decision-making to recommend AAA repair often focuses on 1- to 2-year life expectancy in discussions with patients, against which the annual aneurysm rupture risk is compared.

The purpose of this study was to develop a risk prediction model for mortality during the first year after elective AAA repair. Although the 1-year time threshold is somewhat arbitrary, most would agree that death within 1 year of elective AAA repair indicates ineffective or unnecessary treatment. Such a prediction model could help identify patients who will benefit most from surgery, and potentially those with a short life expectancy in whom medical management would be preferred.

## METHODS

**Subjects and database.** We utilized data collected prospectively by the Vascular Study Group of Northern New England (VSGNNE), a regional cooperative quality improvement initiative developed in 2002 to study regional outcomes in vascular surgery. Further details on this registry have been published previously.<sup>10</sup> Of note, registry data are compared with hospital claims data in regular audits to

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insure reporting of all appropriate cases by participating surgeons.

We included only patients who underwent elective open or endovascular AAA repair of infrarenal aneurysms. Patients with ruptured or symptomatic aneurysms were excluded from this analysis, as were aneurysms that required renal or visceral artery reconstruction. However, pararenal AAAs that required aortic clamping above one or both renal arteries, but with an infrarenal anastomosis, were included.

**Definitions.** Patients were evaluated for pre-existing medical comorbidities including chronic obstructive pulmonary disease (COPD; chart history), congestive heart failure (CHF; chart history or documented ejection fraction <50% on preoperative testing), coronary artery disease (CAD; any history of angina, myocardial infarction [MI], prior coronary intervention, or EKG changes consistent with previous MI), chronic renal insufficiency (CRI; Cr  $\geq$  1.8 mg/dL), end-stage renal disease (ESRD; on dialysis), diabetes mellitus (DM; chart history, designated as diet-controlled, on oral hypoglycemic agents, or on insulin), hypertension (HTN; chart history or blood pressure  $\geq$  140/90), hyperlipidemia (HLIP; chart history), and prior aortic surgery.

Additionally, patients were evaluated for additional preoperative factors including a family history of aneurysm, location of intended aortic clamp site, and perioperative statin, aspirin, and beta-blocker use. Also, operating surgeons for endovascular aneurysm repair (EVAR) patients were asked to prospectively judge whether patients were unfit for an open operation.

**Data collection.** Trained nurses or clinical data abstractors entered data prospectively on over 70 clinical and demographic variables ([www.VSGNNE.org](http://www.VSGNNE.org)). Research analysts were blinded to patient, surgeon, and hospital identity. All patients who underwent AAA repair by 50 participating surgeons in 11 study hospitals from January 1, 2003 to September 30, 2007 were included in the analysis. A current version of the Social Security Death Index as well as 1-year follow-up data obtained as part of VSGNNE were used to confirm the survival status of all patients during the first year after AAA repair.

**Risk model construction.** The primary outcome measure was mortality at 1 year and the secondary endpoint was 30-day mortality. At the time of data analysis, 84% of open patients and 77% of EVAR patients were at least 1 year from their operation. Survival was calculated using life table methodology.

Univariate analysis was performed using a Pearson Correlation Coefficient. Risk factors found by univariate analysis to be associated with 1-year mortality with a *P* value of <0.1 were used in the multivariate Cox proportional hazards model. This model was then used to calculate hazard ratios (HR) and 95% confidence intervals (CI) for postoperative death within 1 year of surgery.

Separate models were constructed for open AAA repair and EVAR patients using factors found in multivariate analysis to predict 1-year mortality. Using Cox-Snell resid-

**Table.** Demographics

	Open <i>n</i> = 748	EVAR <i>n</i> = 639	<i>P</i> value
Mean age (y)	71	74	.001
Male gender	74%	82%	.001
Aneurysm diameter (cm)	6.0 $\pm$ 1.3	5.7 $\pm$ 1.0	<.0001
Comorbidities			
CHF	6%	13%	<.0001
CAD	31%	31%	.89
H/O coronary intervention	29%	31%	.63
COPD	36%	39%	.60
CRI (creatinine $\geq$ 1.8)	8%	7%	.47
HTN	75%	78%	.49
Diabetes mellitus	14%	18%	.17
Diet-controlled	4%	5%	
Oral meds	8%	11%	
On insulin	1%	1%	
Suprarenal clamp	14%	—	—
Deemed "unfit" for open surgery	—	28%	—
Risk factor modification			
Perioperative $\beta$ -blockade	81%	70%	<.0001
ASA/Plavix	65%	68%	.87
Statin	54%	59%	.71
Smoking			
Current (w/i 1 y)	39%	30%	<.0001
Prior (quit >1 y)	47%	57%	.37
Never	8%	12%	.21

EVAR, Endovascular aneurysm repair; CHF, congestive heart failure; CAD, coronary artery disease; COPD, chronic obstructive pulmonary disease; CRI, chronic renal insufficiency; H/O, history of; HTN, hypertension; ASA, aspirin.

uals, predicted 1-year mortality was determined based on each patient's risk factor profile. The predictive ability of each model was then evaluated by generating an observed-to-expected outcome ratio across the range of risk identified.

All analyses were performed using Microsoft Excel (Redmond, Wash) and STATA (College Station, Tex).

## RESULTS

**Patient characteristics.** A total of 1387 patients underwent elective repair of an asymptomatic infrarenal AAA by either an open procedure (*n* = 748) or EVAR (*n* = 639). Patients in the EVAR group tended to be older (74 vs 71 years), had smaller aneurysm diameter (5.7 vs 6.0 cm), and were more likely to have a history of CHF and smoking (Table).

**Mortality.** Thirty-day and 1-year mortality was 2.3% and 5.8% after open repair, and 0.5% and 5.7% after EVAR, respectively (Fig 1). Thirty-day mortality was significantly higher after open AAA repair (*P* < .001), but 1-year mortality was comparable with EVAR (*P* = .91). In the open cohort, there were 43 patient deaths within 1 year of operation of which 17 (40%) occurred within 30 days, and 36 deaths within 1 year of EVAR, with only three (8%) of these within 30 days.

**Open AAA repair.** Univariate predictors for 1-year mortality included age (*P* = .03), COPD (*P* = .002), CRI (*P* < .0001), family history of AAA (*P* = .04), anterior

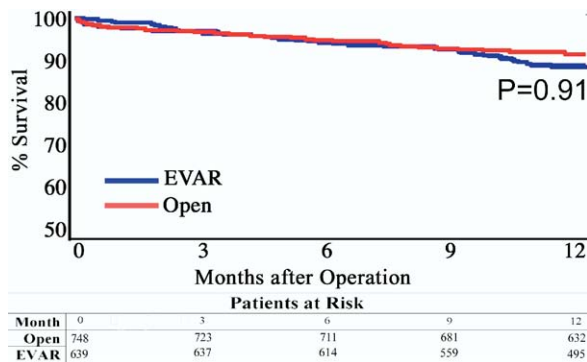


Fig 1. Kaplan-Meier curves demonstrating 1-year survival after open and endovascular aneurysm repair (EVAR).

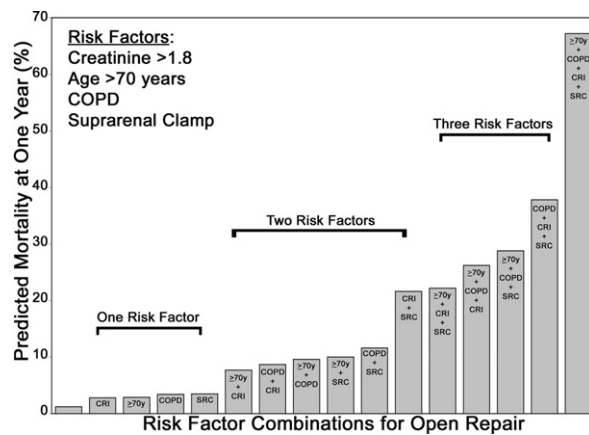


Fig 2. Predicted mortality at 1 year after open aneurysm repair stratified by risk factor combinations.

surgical exposure ( $P = .06$ ), and suprarenal aortic clamp site ( $P < .0001$ ). By multivariate analysis, predictors of 1-year mortality in order of importance included a suprarenal aortic clamp site ( $P < .0001$ ; HR 3.8, 95% CI 1.9-7.5), a history of COPD ( $P < .0001$ ; HR 3.6, 95% CI 1.9-7.0), age  $\geq 70$  ( $P = .007$ ; HR 2.9, 95% CI 1.3-6.3), and CRI ( $P = .008$ ; HR 2.3, 95% CI 1.3-6.2).

Predicted 1-year mortality using the multivariate model ranged from 1.2% in patients with no risk factors present to 67% if all risk factors were present (Fig 2). A model using simply the number of these risk factors present showed good correlation ( $r = .97$ ) between predicted and actual mortality, which ranged from 1.2% to 66% in these groups (Fig 3, A). Actual survival of patients with 0-4 risk factors stratified well by Kaplan-Meier analysis (Fig 4).

**Endovascular AAA repair.** By univariate analysis, age  $\geq 70$  ( $P = .02$ ), CHF ( $P = .007$ ), COPD ( $P = .007$ ), CRI ( $P = .04$ ), and aneurysm diameter  $\geq 6.5$  cm ( $P = .02$ ) were found to be predictive of 1-year mortality after EVAR. Also, a designation of unfit for open operation, which was given to 28% of EVAR patients ( $n = 182$ ), was predictive of 1-year mortality ( $P = .056$ ).

By multivariate analysis, however, only a history of CHF ( $P = .002$ ; HR 3.2, 95% CI 1.6-6.5) and aneurysm size  $\geq 6.5$  cm ( $P = .04$ ; HR 2.2 95% CI 1.0-4.8) were predictive of 1-year mortality after EVAR. Patients without CHF and an aneurysm greater than 6.5 cm had a predicted mortality at 1 year of 3.6%, while those with both risk factors had a predicted mortality of 23% at 1 year. The observed-to-expected ratio was excellent for this model at 0.96 (Fig 3, B).

**Diameter of AAA at time of repair.** Aneurysm diameter at the time of repair was evaluated for each risk group. As the number of risk factors increases, the diameter at repair increased significantly. Mean aneurysm diameter for patients with no independent risk factors for mortality within 1 year was 5.6 cm. Those patients with three or four independent risk factors had a mean aneurysm diameter of 6.7 cm, which was statistically larger than the no risk factor group ( $P < .0006$ ) (Fig 5).

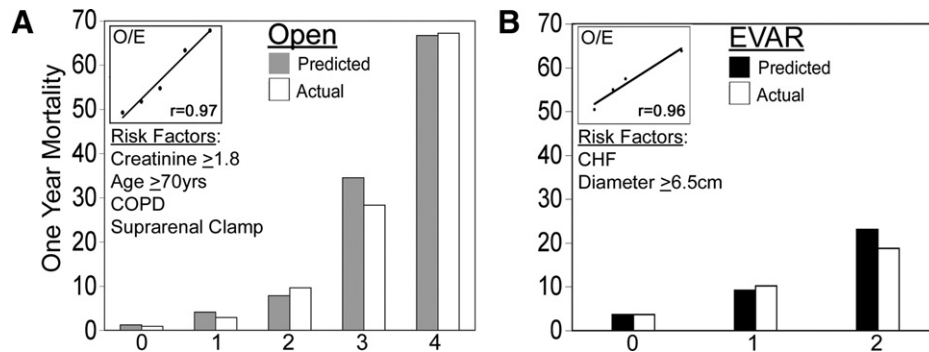
## DISCUSSION

Numerous publications have found medical comorbidities that are independent predictors of in-hospital or 30-day mortality after open AAA repair, including CHF, CRI, COPD, CAD, advanced age, and aneurysm diameter.<sup>6,11,12,13-17</sup> Long-term (usually 5 years) mortality risk assessment models have been developed using similar risk factors with variable degrees of success.<sup>3,4,7</sup> For EVAR, Barnes and colleagues demonstrated that aneurysm diameter was independently predictive of perioperative mortality, and this along with CRI, age, and ASA (American Society of Anesthesiologists) score were predictive of 3-year and 5-year mortality after EVAR.<sup>18</sup> Others have also demonstrated aneurysm diameter, age, and CRI to be independent predictors of late death after EVAR.<sup>19,20</sup>

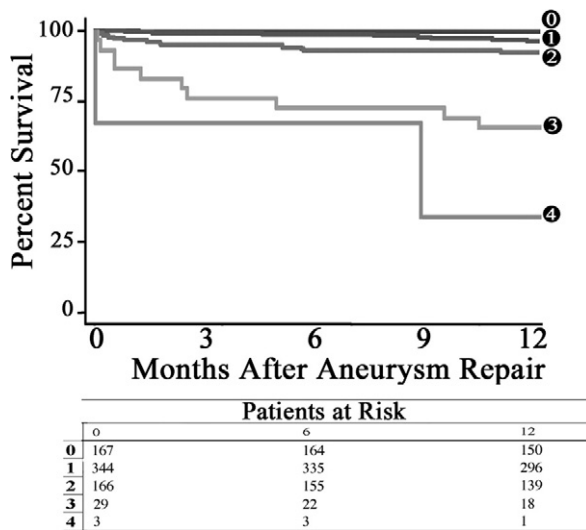
In our patient population, less than half of the deaths that occurred within the first year after open operation occurred within 30 days. For the EVAR group, this number is even more striking with just 8% of deaths within 1 year attributable to 30-day mortality. This emphasizes the importance of using longer survival (at least 1 year) to assess the overall benefit of AAA repair, even though most surgeons focus on in-hospital results.

One-year mortality was selected for analysis to provide a useful counterpoint to the often cited annual AAA rupture risk when discussing decision making with patients. Further, we reasoned that most patients and surgeons would judge death within 1 year of AAA repair to represent ineffective treatment, unless an aneurysm was very large with very high rupture risk. Finally, convalescence time for operative recovery, especially for open repair, occupies a significant proportion of the first year after surgery, making longer survival even more important. For these reasons, we developed a model to predict 1-year mortality.

For open AAA repair, our analysis of 1-year mortality yielded a highly predictive risk assessment model. Age  $\geq 70$  years, CRI, COPD, and a suprarenal clamp requirement were all found to be independent predictors of mortality and are available preoperatively. Although suprarenal clamp



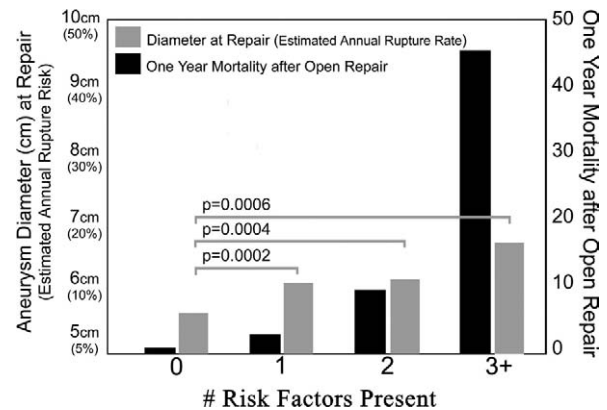
**Fig 3.** Predicted and actual 1-year mortality rates based on the number of risk factors identified after open abdominal aortic aneurysm (AAA) repair (A) and endovascular aneurysm repair (EVAR) (B).



**Fig 4.** Kaplan-Meier curve demonstrating 1-year survival after open aneurysm repair stratified by risk factor groups.

site has been variably associated with operative mortality in previous reports,<sup>21-25</sup> in our patient population this was an important predictor of 1-year mortality (and also 30-day mortality, data not shown). This was especially true when suprarenal clamp site is combined with other medical comorbidities (Fig 2). It is unclear why suprarenal clamp site correlates with 1-year mortality. In part, this is related to postoperative mortality, but 53% of patients who had a suprarenal clamp and who died within 1 year did so after the initial 30 days. It is possible that the need for suprarenal clamping is a surrogate marker for more advanced disease, or perhaps correlates with other operative complications such as renal failure, which might lead to higher mortality rates. Additionally, although suprarenal clamp site is usually chosen preoperatively based on axial imaging, it is somewhat subject to surgeon's preference and can also change intraoperatively due to unforeseen technical challenges.

Patients with none of the above risk factors had a 1-year mortality of only 1.2% after open repair, while those pa-



**Fig 5.** AAA diameter at time of repair (and estimated annual rupture risk) (gray bars) compared with predicted 1-year mortality after open abdominal aortic aneurysm (AAA) repair (black bars) as a function of number of risk factors present for mortality after open repair, and rupture risk as a function of diameter estimated by the authors based on relevant literature.<sup>30-33</sup>

tients with three or four risk factors had predicted mortality rates ranging from 22% to 67% at 1 year. This would suggest that patients with few of these risk factors are good candidates for surgery at smaller aneurysm diameters. In contrast, patients with several of these risk factors have a substantially higher 1-year mortality after open operation, suggesting that repair should only be performed at larger AAA diameter, unless EVAR can be performed.

Although rupture risk based on aneurysm diameter is somewhat imprecise, an accepted estimate for rupture risk in patients with 5 to 5.9 cm aneurysms in the absence of rapid expansion is  $\sim 6.5\%$  per year.<sup>26</sup> Current general practice is to repair aneurysms  $\geq 5.5$  cm. In a high-risk patient with three or four risk factors, the high risk of mortality within 1 year of elective open AAA repair substantially exceeds the rupture risk of most 5.5 cm AAAs. Interestingly, when patients undergoing open repair were stratified by aneurysm diameter at repair, it appears that surgeons are taking these risk factors into account since patients with higher risk for 1 year mortality had larger diameters at time



of repair (Fig 5). When estimated annual rupture risk (Fig 5, *gray bars*) is compared with predicted 1-year mortality for each risk factor group (Fig 5, *black bars*), it appears that patients with multiple risk factors perhaps should have much larger aneurysms before open repair in order to justify the risk of operation. In contrast, patients with none to two risk factors appear to have a favorable ratio of higher estimated rupture risk compared with lower estimated 1-year mortality (Fig 5). These risk prediction models have not yet been validated against a separate database, but will of course be more valuable once this is done. In addition, it is possible that future analyses of more patients will disclose other significant predictors of outcome. Finally, decision making for patients must always be individualized, using prediction factors to inform, but not to make decisions.

In contrast to open repair, perioperative mortality after EVAR is rare, even in relatively high-risk patients,<sup>9,27-29</sup> but large series have demonstrated short- and long-term predictors of survival.<sup>1,18,19</sup> In our analysis, the 30-day mortality rate for EVAR patients was only 0.5%. By 1 year, however, mortality after EVAR reached 5.7%, which was equivalent to that for open repair. A number of risk factors were found to be predictive of 1-year mortality in univariate analysis, including CHF, COPD, age, and aneurysm diameter  $\geq 6.5$  cm. However, only CHF and aneurysm diameter  $\geq 6.5$  cm were independent predictors of 1-year mortality by multivariate analysis. CHF as a single predictor for mortality after EVAR had a greater impact on survival than any single risk factor for mortality after open AAA repair based on the hazard ratios. The other independent predictor, aneurysm diameter  $\geq 6.5$  cm also increases rupture risk, making this variable less helpful in decision making, since it argues both for and against repair. Creation of a more comprehensive prediction model for EVAR was not possible in our study, likely due to a small number of patients who died within 1 year and interaction between the risk factors that we identified in the univariate analysis.

Currently, AAA repair is recommended when the aneurysm rupture risk exceeds the operative risk. These data demonstrate that certain patients are at high risk for aneurysm repair, especially by open surgical methods. Embracing the concept of 1-year survival as a measure of treatment success can facilitate clinical decision-making before AAA repair.

## AUTHOR CONTRIBUTIONS

Conception and design: AB, PG, BN, JC  
Analysis and interpretation: AB, PG, BN, DL, JC  
Data collection: DL, JC  
Writing the article: AB, PG, JC  
Critical revision of the article: AB, PG, BN, DL, JC  
Final approval of the article: AB, PG, BN, DL, JC  
Statistical analysis: AB, PG, BN, DL  
Obtained funding: JC  
Overall responsibility: JC

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## DISCUSSION

**Dr Peter Glaviczi** (Rochester, Minn). I enjoyed your paper very much and would like to congratulate you on an in-depth analysis of data of mortality at 1 year. You mentioned that you included 49 surgeons of 11 centers, and what I wanted to hear is if your data depended on surgeon's expertise. What has happened is that more recently we do less open repairs in more complex patients. We do more suprarenal or supraceliac clamping because most of the usual bread-and-butter infrarenal aneurysms are repaired endovascularly. So I am wondering if surgeon's experience is important or not in the outcome.

When we looked recently at results of 126 juxtarenal aneurysms, we found that the level of aortic clamp replacement (suprarenal vs suprarenal: supravisceral vs inter-renal) had no significant effect on the development of cardiac, pulmonary, or renal complications nor on mortality. I am wondering if you found a difference or whether you had enough patients who had supraceliac clamping who maybe had a higher mortality.

Otherwise your 1-year outcome data are very similar to ours, we had a 6% mortality by 1 year after juxtarenal aneurysm repair and a 0.8% mortality at 30 days, which is very similar to our results with infrarenal aortic aneurysm.

**Dr Adam W. Beck.** Although we have the capability of looking at surgeon effect, we did not do so for this particular study. I saw your publication regarding juxtarenal aneurysms. We included all patients who had an aortic clamp above both renal arteries, but we did not differentiate suprarenal vs supraceliac. If we looked at just a clamp above one renal artery, those patients had a similar outcome to the infrarenal clamp group in our database.

**Dr Jack Cronenwett.** When we look at these data, we find some apparent impact of surgeon volume, but the numbers become very small and impossible to accurately analyze yet in multivariate analysis. We are using the registry primarily for quality improvement efforts. But overall, the 30-day mortality results in both the open and endovascular groups were quite excellent.

**Dr Tara Mastracci** (Cleveland, Ohio). Thank you for your very thoughtful analysis. This is an area of interest for us at the Cleveland Clinic as well. I was wondering about your choice of a 1-year endpoint. In an era where we are now achieving very acceptable perioperative outcomes for endovascular repair even in high-risk patients who have multiple comorbidities threatening their longevity, should we be focusing on long-term outcomes,

instead of 1-year mortality, to better inform the decision to operate on only the high-risk patients in whom we can actually prolong survival?

**Dr Beck.** This is an important point. If you look at most published risk prediction models for AAA surgery, they look primarily at perioperative mortality. There are also a number of publications that have identified independent risk factors for mortality at 3 and 5 years, but no risk prediction models. Further, many other factors such as subsequent cancer or heart disease affect 5-year mortality. We chose the 1-year time point because it corresponds well with 1-year rupture risk, which is often discussed with patients during decision making.

**Dr John Hallett** (Charleston, SC). This is really an important study because the methodology that you are using for vascular has been used to improve cardiac care in your region of the country. And, I am interested in how you are going to use this information now with the surgeons to improve care. Are you going to use this methodology in a prospective way so the surgeon can advise the patient? Are you going to use it to advise the surgeon in decision-making? How are you going to use these data in quality improvement that has been used so well for cardiac surgery?

**Dr Beck.** We presented these data at the recent meeting of the Vascular Study Group and it was believed to be important for future clinical decision making. To facilitate this, it was decided to make small pocket cards so VSGNNE members could easily remember these risk factors.

**Dr Maciej Dryjski** (Buffalo, NY). What is striking for me is that congestive heart failure (CHF) is a predictive factor for mortality in the endovascular group but not in the open group. It seems to me that we perhaps do something different when we treat endovascularly a patient with an aneurysm. The question is if the dye given in perioperative evaluation, during the operation, and then on the follow-up CTs, has an influence on 1-year postoperative mortality?

**Dr Beck.** We did look at the amount of contrast that patients received during EVAR in this study. We were trying to look at preoperative data so that it could help the surgeon and the patient make decisions before the operation. But in other analyses, we have not found that contrast volume affected mortality after EVAR.

There were more patients in the EVAR group that had CHF by percentile, and there were more patients that had more severe

CHF in the EVAR group. This may be why CHF was an independent predictor of outcome in the EVAR patients and not in the open repair patients.

**Dr Jerry Goldstone** (Cleveland, Ohio). One of the issues that you did not discuss was gender differences. Were there any differences male vs female, and in particular, aneurysm size? There has been some suggestion that women's aneurysms may rupture at a smaller size and, therefore, the threshold for operating might be lower than what we.

**Dr Beck.** We did look at gender. There was no difference in outcomes between men and women, although your point is well taken about aneurysm size and rupture risk. Certainly we cannot prescribe fixed diameter thresholds for elective repair. However, for patients with three to four identified risk factors for mortality after open repair, it would appear that repair should be delayed until a larger diameter is reached. On a patient-by-patient basis, you definitely would have to consider gender in setting the optimal diameter threshold for elective repair.